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# Shock Index as Simple Clinical Independent Predictor of In-hospital MACEs in NSTEMI Patients Presenting with Heart Failure

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**Background:** Identification of Non-ST Elevation Myocardial Infarction (NSTEMI) patients at higher risk of in-hospital complications is very important. Such identification will give crucial information in determining treatment strategy especially for those come with heart failure. One of the simple predictor for short term prognosis in acute coronary syndrome is shock index (SI), which is the ratio of heart rate over systolic blood pressure on admission. There had not been any study conducted to evaluate the use of SI in NSTEMI patients come with heart failure. The aim of this study is to evaluate the SI compared with other routine clinical and laboratory examination as a predictor of in-hospital major adverse cardiac events (MACEs) in NSTEMI patients presenting with heart failure.
**Methods:** We performed a retrospective analysis of NSTEMI patients with heart failure admitted to Haji Adam Malik General Hospital in Medan from January 2014 until July 2015. SI was calculated as the ratio of heart rate over systolic blood pressure on presentation. Patients presenting with cardiogenic shock were excluded.
**Results:** There were 55 patients eligible in this study. In-hospital MACEs was found in 24 patients (44%) compared with 31 patients (56%) without in-hospital MACEs. Patients with in-hospital MACEs were older (60.6±10.8 vs. 57.2±7.9, p=0.178), had less history of dyslipidemia [8(33%) vs. 19 (61%), p=0.032], faster heart rate (111.4±35.8 vs. 96.5±24.3, p=0.032], higher GRACE score [139(98-187) vs. 120 (91-148); p=0.001], and higher SI [0.83(0.57-1.5) vs. 0.67 (0.38-1.27), p=0.013). SI >0.8 was the only independent predictor of MACEs in NSTEMI patients presenting with heart failure (OR=4.3, CI=1.247-14.328, p=0.048).
**Conclusion:** Beyond other routine examinations, SI is the only independent predictor of in-hospital MACEs in NSTEMI patients presenting with heart failure.

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**Keywords:** NSTEMI, shock index, in-hospital MACEs

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Penelitian Klinis

# Indeks Syok sebagai Prediktor Klinis Sederhana terhadap Kejadian Kardiovaskular Mayor Selama Perawatan pada Pasien IMANEST dengan Gagal Jantung

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**Latar Belakang:** Identifikasi terjadinya komplikasi selama perawatan pada pasien dengan Infark Miokard Akut Non-elevasi Segmen ST (IMANEST) merupakan hal yang sangat penting dilakukan. Hal ini untuk menentukan strategi pengobatan selanjutnya pada kelompok pasien ini terutama pada pasien yang tiba dengan kondisi gagal jantung. Salah satu pengukuran terbaru dalam memprediksi kejadian kardiovaskular mayor (KKvM) yang sederhana adalah pengukuran indeks syok (IS), yakni pembagian laju denyut jantung terhadap tekanan darah sistolik pada saat tiba di rumah sakit. Belum ada penelitian sebelumnya yang menilai manfaat IS dalam memprediksi komplikasi pasien IMANEST dengan gagal jantung. Tujuan penelitian ini adalah melihat IS dan membandingkannya dengan pemeriksaan klinis dan laboratorium rutin lainnya dalam prediksi terhadap KKvM selama perawatan pada pasien IMANEST dengan gagal jantung.
**Metode:** Penelitian retrospektif terhadap pasien IMANEST yang tiba di Rumah Sakit Umum Pusat Haji Adam Malik (RSUP HAM) dengan kondisi gagal jantung pada periode Januari 2014 hingga Juni 2015. IS dihitung dengan pembagian laju denyut jantung terhadap tekanan darah sistolik saat tiba. Pasien dengan syok kardiogenik dieksklusikan.
**Hasil**: Terdapat 55 pasien yang memenuhi kriteria inklusi dan eksklusi pada penelitian ini. Pasien dengan KKvM sebanyak 24 orang (44%) dan tanpa KKvM 31 orang (56%). Pasien yang mengalami KKvM terlihat lebih tua (60,6±10,8 vs. 57,2±7,9; p=0,178), dengan riwayat dislipidemia lebih sedikit [8(33%) vs. 19 (61%), p=0.032], laju denyut nadi lebih cepat (111,4±35,8 vs. 96,5±24,3; p=0,032], skor GRACE lebih tinggi, [139(98-187) vs. 120 (91-148); p=0,001], dan nilai IS lebih tinggi [0,83(0,57-1,5) vs. 0,67 (0,38-1,27); p=0,013). Nilai IS >0,8 merupakan prediktor independen satu-satunya terhadap KKVM selama perawatan (OR=4,3; IK=1,247-14,328; p=0,048).
**Kesimpulan**: Dibandingkan dengan pemeriksaan rutin lainnya, pemeriksaan IS merupakan prediktor KKvM satu-satunya selama perawatan pada pasien IMANEST yang tiba dengan kondisi gagal jantung.

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**Kata kunci:** IMANEST, indeks syok, KKVM

## Introduction

Coronary artery disease (CAD) including acute coronary syndromes (ACS), is the most prevalent manifestation of cardiovascular disease and is associated with high mortality and morbidity.[1] These are currently the leading cause of death in industrialized countries and

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are expected to become so in emerging countries by 2020.[1,2]

Non-ST Elevation Myocardial Infarction (NSTEMI) patients appear to have lower short-term mortality compared with ST Elevation Myocardial Infarction (STEMI) individuals, but both have similar outcomes in a long term.[3] NSTEMI patients have differences in baseline characteristics, including older age and a greater prevalence of co-morbidities.[3-6] The clinical spectrum of non-ST-elevation ACS (NSTE-ACS) may range from patients free of symptoms at presentation to individuals with ongoing ischemia, heart failure, electrical or hemodynamic instability or cardiac arrest.[3] These wide clinical spectrum of NSTEMI require physician to perform good risk stratification. Given that risk stratification is a starting point in determining whether a patient with NSTEMI will undergo an invasive or conservative strategy, attempts to improve predictive ability of the patient outcome are essential.

Currently, Global Registry of Acute Coronary Events (GRACE) score is the most accurate score for predicting both short-term and long-term risks.[7,8] Eight parameters are used for calculating GRACE score that include patient's age, heart rate, systolic blood pressure, Killip class, serum creatinine level, cardiac arrest at hospital admission, ST-segment deviation in ECG and elevated cardiac marker.[9] This score are sophisticated and one must calculate this score using smartphone, tablet or laptop.[10]

NSTEMI patients can present with stable conditions, heart failure, or cardiogenic shock. Patients with Killip class II and III (heart failure) and IV (cardiogenic shock) made up 11% of the population but accounted for 30% of the deaths.[11] In Killip IV mortality is high (67%) and guidelines had specific mandates for physician to perform early invasive strategy.[3] In Killip II-III, mortality is ranging from 17%-38% and risk stratification must be done accurately.[11] Data from the GRACE registry shows 13% of ACS patients had an admission diagnosis of heart failure (Killip class II or III). Heart failure (HF) on admission was associated with a marked increase in mortality rates during hospitalization and at 6 months after discharge.[12]

In the GRACE score, the Killip class is one of the components of the score. This becomes the limitation of the GRACE score to predict the complication other than mortality in Killip class II, III, and IV.

One of the emerging and simple predictor of cardiovascular complications in ACS is the shock index (SI).[13-16] The SI parameter defined as the ratio of heart rate (beats per minute) to systolic blood pressure (millimeters of mercury) at admission with the normal value range from 0.5-0.7.[17] SI will rise in conditions where heart rate (HR) increases while the systolic blood pressure (SBP) decreases such as hypovolemia and left ventricle systolic dysfunction.[18]

In AMI, there will be myocardial necrosis followed by contractility dysfunction that can lead to heart failure and cardiogenic shock. Neuro-hormonal activation will occur to compensate that occurrence. The neuro-hormonal compensation involves the sympathetic system, renin-angiotensin-aldosterone (RAA) system, and the release of antidiuretic hormone. Such compensation reflects in the increasing of HR and SBP.[19] Over-activity of sympathetic system correlates with severity of left ventricle dysfunction.[20,21] In the pathophysiology view, SI is an integrative parameter reflecting hemodynamic conditions of patients.[15] Therefore, SI may be useful in predicting prognosis in NSTEMI patients with heart failure.

A few studies have shown that SI is a predictor of major adverse cardiac events (MACEs) in ACS, AMI, STEMI and NSTEMI. However, there is only one study on NSTEMI which performed by Kobayashi et al.[15] The study showed that SI ≥0.7 had higher in-hospital mortality, higher cardiogenic shock at admission and lower left ventricle ejection fraction (LVEF).[15]

SI seems to be a simple but very useful tool in predicting AMI complications. This examination can be performed in situations where laboratory value cannot be obtained. SI also can be used as a bedside tool in daily practice. Hence, this examination is very useful in developing countries such as Indonesia.

The aim of this study is to evaluate the SI compared with other routine clinical and laboratory examination as a predictor of in-hospital major adverse cardiac events (MACEs) in NSTEMI patients presenting with heart failure.

## Methods

This is a retrospective study of NSTEMI patients who were treated at the National General Hospital Adam Malik from January 2014 to July 2015. Subjects are patients with NSTEMI diagnosis based on the criteria from Indonesian Heart Association (IHA) with complaints of acute angina pectoris without

ST segment elevation persistent in two contiguous leads with significant increasing cardiac enzymes[22] and presenting signs defined in Killip II and/or III classification. Killip Class II describes individuals with findings of mild to moderate heart failure (S3 gallop, rales below half-way up lung fields or elevated jugular venous pressure). Killip Class III describes individuals with occurrence of pulmonary edema.

Exclusion criteria in this study are patients with cardiogenic shock, severe bradycardia resulting from 2nd-3rd degree AV block, left branch bundle block (LBBB) when an unknown previous ECG and patients with severe comorbidities at admission i.e. sepsis, severe exacerbating chronic obstructive pulmonary disease (COPD), acute renal failure requiring emergency hemodialysis, and acute stroke that could influence the occurrence of major adverse cardiac events (MACEs).

The MACEs is defined for all-cause mortality and cardiogenic shock in all of the patients. In patients with Killip II, MACEs is defined by previously mentioned criteria plus the occurrence of acute pulmonary edema during hospitalization.

According to the formula for computing sample size for categorical analysis in independent samples, we found the minimal number of samples is 21 for each group. Our samples were collected from January 2014 until July 2015.

SI was calculated as the ratio of HR/SBP on admission. We used the cut-off value of 0.8 based on the previous study.[13]

We searched the medical records of NSTEMI patients with Killip II and III in this periode and divided them into groups of patients with MACEs and without MACEs. Confounding variables were also noted, including age, gender, risk factors for coronary heart disease (hypertension, diabetes, hypercholesterolemia, smoking, and age), blood pressure, heart rate, Killip class, routine laboratory findings (complete blood count, renal function, electrolytes, initial blood glucose, and cardiac enzymes), arrhythmias, treatment and medication for the patients. We noted the clinical course of patients during treatment to see in-hospital MACEs afterward.

## Statistical Analysis

Significant statistical difference was defined as p value <0.05. Categorical variables were presented with the number or frequency (n) and percentage (%). Numerical variables were presented with a mean (average) and standard deviation for normally distributed data, and using median if the data were not normally distributed. Normality test was done using one sample Kolmogorov-Smirnov (n >50) or the Shapiro Wilk (n <50). Normally distributed data were analyzed with two independent-samples T-Test and Mann Whitney U test for the data that were not normally distributed. Categorical variables were analyzed using chi square or Fisher tests. Variables found to be significant on bivariate analysis test were continued into multivariate analysis with logistic regression test.

## Results

During the period from January 2014 to July 2015 there were 60 patients admitted with a diagnosis of NSTEMI dan HF (Killip II and III). From these 60 patients, 55 patients met the inclusion and exclusion criteria.

Baseline characteristics are shown in **Table 1**. Subjects are divided into two groups based on occurrence of MACEs. The groups with MACEs were older, more in Killip III conditions, but were not significantly different. In this study, patients with in-hospital MACEs had faster heart rate (111.4±35.8 vs. 96.5±24.3, p=0.032), fewer history of dyslipidemia [8 (33%) vs. 19(61%), p=0.040], higher shock index [0.83 (0.57-1.5) vs. 0.67 (0.38-1.27), p=0.013], and higher GRACE score [139 (98-187) vs. 120 (91-148), p=0.001)]. There were no statistical significant differences in laboratory examination and other CAD risk factor beside history of dyslipidemia.

MACEs occurred in 24 patients. All-cause mortality found in 11 patients (20%), CS in 5 patients (9%), and acute pulmonary edema in 8 patients (15%).

From the bivariate analysis, we found two variables that had significant differences, which was SI >0.8 and history of dyslipidemia. We include 5 variables that had p value <0.25 (history of hypertension, GFR <30 mL/min/1.73 $m^2$, GRACE score >140. Killip class III, and Chloride level <97) plus one variable HR>100 bpm from author consideration based on previous study.[23] These eight variables then entered into multivariate analysis. The results showed shock index >0.8 become the independent predictor of MACEs from the multivariate analysis [OR 4.226 (1.247-14.328); p=0.021].

**Table 1.** Baseline characteristics.

| | Overall n=55 | In-hospital MACE (+) n=24 (44%) | In-hospital MACE (-) n=31 (56%) | p value |
|---|---|---|---|---|
| Age (years) | 58.7±9.3 | 60.6±10.8 | 57.2±7.9 | 0.178* |
| Sex (male) | 40 (73%) | 19 (79%) | 21 (68%) | 0.345** |
| Body Mass Index (BMI) | 24.5 (20.3-35) | 24.4 (20.8-35.2) | 24.5 (20.3-32) | 0.905*** |
| Risk factor | | | | |
| Hypertension | 41 (75%) | 16 (67%) | 25 (81%) | 0.238** |
| Diabetes | 24 (43%) | 10 (42%) | 14 (45%) | 0.796** |
| Dyslipidemia | 27 (49%) | 8 (33%) | 19 (61%) | 0.040** |
| Smoker | 36 (66%) | 15 (63%) | 21 (68%) | 0.685** |
| Hemodynamic conditions | | | | |
| Systolic BP | 134.7±29.0 | 131.3±32.7 | 137.4±25.9 | 0.439* |
| Diastolic BP | 80 (50-130) | 80 (60-120) | 80 (50-130) | 0.661*** |
| Heart rate | 102.9±25.8 | 111.4±25.8 | 96.5±24.3 | 0.032* |
| Atrial Fibrillation (n, %) | 3 (5%) | 3 (12.5%) | 0 (0%) | 0.077** |
| Shock Index | 0.75 (0.38-1.5) | 0.83 (0.57-1.5) | 0.67 (0.38-1.27) | 0.013*** |
| SI >0.8 | 20 (36%) | 13 (54%) | 7 (23%) | 0.016** |
| Killip Class III | 17 (31%) | 10 (42%) | 7 (23%) | 0.129** |
| Presenting with infection | 14 (25.5%) | 8 (33.3%) | 6 (19.4%) | 0.238** |
| Laboratory findings | | | | |
| Hemoglobin (mg/dl) | 12.8±2.2 | 12.7±1.9 | 12.9±2.4 | 0.627* |
| Leucocyte (/mm$^3$) | 12110 (6280-33990) | 11765 (6280-26500) | 13782 (6320-33990) | 0.879*** |
| Admission blood glucose level (mg/dl) | 141 (59-708) | 160 (70-708) | 138 (59-423) | 0.290*** |
| Ureum (mg/dl) | 55.1 (15-345) | 53.6 (15-260) | 56.8 (21.9-345) | 0.728*** |
| Creatinine (mg/dl) | 1.5 (0.6-12) | 1.54 (0.69-12) | 1.4 (0.6-8.4) | 0.435*** |
| GFR (mL/min/1.73 m2) | 47.8±24.6 | 44.9±27.3 | 49.6±22.9 | 0.546* |
| GFR <30 mL/min/1.73 m2 | 13 (23%) | 8 (33%) | 5 (16%) | 0.136** |
| Natrium (mEq) | 136 (118-145) | 134±7.1 | 136±4.6 | 0.225* |
| Potassium (mEq) | 4.1 (2.6-6) | 4.1 (3.2-6) | 4.1 (2.6-6.2) | 0.363*** |
| Chloride (mEq) | 105 (86-111) | 103 (86-110) | 105 (94-114) | 0.104*** |
| Troponin T (ng/ml) | 0.45 (0.1-2) | 0.58 (0.11-2) | 0.37 (0.1-2) | 0.257*** |
| Osmolality (osm/L) | 290.3 (255.2-353.3) | 291±15 | 292±13 | 0.557* |
| GRACE score | 131.3±22.9 | 139 (98-187) | 120 (91-148) | 0.001*** |
| GRACE risk score >140 | 21 (38%) | 12 (50%) | 9 (29%) | 0.112** |
| In-hospital medication and treatment | | | | |
| Diuretics | 55 (100%) | 24 (44%) | 31 (56%) | NS |
| ACE-inhibitors or ARBs | 36 (65.4%) | 15 (62.5%) | 21 (67.8%) | 0.685** |
| Beta blockers | 31 (56.3%) | 13 (54.2%) | 18 (58.1%) | 0.773** |
| Double antiplatelet | 51 (92.7%) | 21 (87.5%) | 30 (96.7%) | 0.307** |
| Anticoagulant | 51 (92.7%) | 21 (87.5%) | 30 (96.7%) | 0.307** |
| Coronary Angiography | 17 (30.9%) | 6 (25%) | 11 (35.5%) | 0.404** |
| Angiography Result | | | | |
| 3VD and/or LM | 14 (25.4%) | 6 (25%) | 8 (25.8%) | |
| 2 VD | - | - | - | NS** |
| 1 VD | 3 (5.5%) | - | 3 (9.7%) | |
| Revascularization | 3 | 1 | 2 | NS** |

* Independent T-Test
**Chi Square
***Mann Whitney

**Table 2**. Bivariate analysis.

| | OR (CI) | p value* |
|---|---|---|
| History of Dyslipidemia | 0.316 (0.104-0.963) | 0.04 |
| History of Hypertension | 0.480 (0.140-1.643) | 0.238 |
| GFR <30 mL/min/1.73 m2 | 2.602 (0.723-9.344) | 0.136 |
| GRACE >140 | 2.444 (0.802-7.449) | 0.112 |
| Killip Class III | 2.449 (0.761-7.885) | 0.129 |
| Heart Rate >100 bpm | 1.700 (0.579-4.989) | 0.333 |
| Chloride <97 mEq | 3.816 (0.671-21.715) | 0.220 |
| SI >0.8 | 4.052 (1.266-12.970) | 0.016 |

*Chi Square

**Table 3**. Multivariate analysis.

| | OR (CI) | p value |
|---|---|---|
| SI >0.8 | 4.226 (1.247-14.328) | 0.021 |
| History of Dyslipidemia | 0.301 (0.092-0.988) | 0.048 |

## Discussions

In this study, patients with in-hospital MACEs had faster heart rate (111.4±35.8 vs. 96.5±24.3, p=0.032). This is the same result with the previous study.[23] This founding suggest that in HF patients, neuro-hormonal compensation as reflected by HR can reflect the clinical severity of the patients thus the complication and outcome.

Many factors can affect heart rate and/or blood pressure and as well as the shock index. The factors such as previous medication that lowering heart rate (eg. beta blocker, digoxin, and non-dihydropyridine), AV block, treatment on antihypertension, and condition such as infection and hypovolemia. From the medical records, we did not find complete and specific notes about previous medication so we cannot put this on the current analysis. As we mentioned above that sepsis patients and severe AV block at admission were excluded so these would not affect our analysis on shock index. There were 9 patients suffered from infection, namely pneumonia (8 patients) and lung tuberculosis (1 patient). However, these condition did not give statistical differences on shock index (p=0.261). There were no patients in hypovolemic state requiring rehydration at admission.

Both SBP and DBP (diastolic blood pressure) did not differ significantly. This finding seems contrary with the results of Bangalore et al study which found the paradox of blood pressure (BP) in predicting mortality in NSTE-ACS.[24] There are few differences in our study which exclude the unstable angina pectoris (UAP) patients, Killip I and IV NSTEMI patients. This finding suggests that in NSTEMI patients presenting with heart failure, BP alone cannot predict the outcome of patients.

From the study of Kobayashi et al[15] patients with SI ≥0.7 had a lower LVEF, higher rate of cardiogenic shock on admission, and higher in-hospital mortality. The study did not specify the patients. Our study specify the patients into the NSTEMI with HF condition. Our study showed that SI >0.8 was the only independent predictor of MACEs. From the statistical analysis, SI is better than BP and HR alone. This finding suggested the role of SI as an integrative hemodynamic parameter.

In this study, Killip class III did not show differences in predicting mortality. This finding is important that between patients in Killip II and III, physician still should be aware of the risk of complications in both group.

Other factors can affect the occurrence of MACEs such as arrhythmias and in-hospital treatment. There were 3 patients presenting with atrial fibrillation and none with neither supraventricular nor ventricular arrhythmias. Yet, there were no significant differences on MACEs.

All of the patients received standard medication for acute coronary syndrome, including double antiplatelet (DAPT), anticoagulant, and statin. There were 3 cases where the DAPT and anticoagulant were stopped due to bleeding. The use of angiotensin converting enzyme inhibitor (ACE-i) and or angiotensin receptor blocker (ARB) was 65% in all patients, and the use of beta blockers (BB) was 56%. When we analyzed the influences of medication and treatment including revascularization to the MACES, we did not found any differences.

Our study showed that patients with in-hospital MACEs had higher GRACE score [139 (98-187) vs. 120 (91-148)] but when divided by the cut-off value of 140, it did not show differences. This finding suggests that GRACE score with the validated cut-off value cannot be used in predicting complications more than just death.

### Limitations of study

This was a retrospective study on secondary data with

small sample size in single center which became one of the limitations of the study. Some of the patients may come with previous medication that affects the HR such as beta blocker, digoxin, non-dihydropyridine calcium channel blocker and the other antihypertension therapy. Unfortunately, in this study we did not have complete data about their medication thus we cannot see the effect. However, the mean value of HR in this study is 102.9 suggesting that all patients maybe were in decompensation state of HF.

Our study found that DBP did not differs significantly. Other model of shock index is the modified shock index (MSI) which is the ratio of HR/mean arterial pressure (MAP). In this study we did not analyze the role of MSI.

Another limitation of this study is that we only performed analysis that compares simple routine clinical and laboratory examination, as such we did not compare with echocardiography parameter such as LVEF. We also did not analyze the effect of revascularization for in-hospital MACEs. This was because only a small percentage of patients were performed coronary angiography (17 patients, 31%).

**Table 4.** Effect of infection on shock index.

| | SI >0.8 (n=20) | SI ≤0.8 (n=35) | p value |
|---|---|---|---|
| Presenting with infection | 5 (25%) | 4 (11.4%) | 0.261* |

*Chi square

## Conclusion

In this study, beyond other routine examination SI was the only independent predictor of in-hospital MACEs in NSTEMI patients presenting with heart failure. SI is a quick, easy, and cheap way to predict in hospital MACEs in NSTEMI patients presenting heart failure but requires better research method and design, also the bigger sample size before it is ready for primetime use. This simple measurement may have important and beneficial role in predicting prognosis in remote and rural area.

## Abbreviations

ACE-i: angiotensin converting enzyme inhibitor
ACS: acute coronary syndromes
ARB: angiotensin receptor blocker
BP: blood pressure
CAD: coronary artery disease
COPD: chronic obstructive pulmonary disease
DBP: diastolic blood pressure
ECG: electrocardiogram
GRACE: global registry of acute coronary events
MACEs: major adverse cardiac events/*kejadian kardiovaskular mayor (KKvM)*
HF: heart failure
HR: heart rate
IHA: Indonesian Heart Association
LBBB: left branch bundle block
LVEF: left ventricle ejection fraction
MAP: mean arterial pressure
MSI: modified shock index
NSTE-ACS: non-ST-elevation ACS
NSTEMI: non-ST elevation myocardial infarction/*infark miokard akut non-elevasi segmen ST (IMANEST)*
RAA: renin-angiotensin-aldosterone
SBP: systolic blood pressure
SI: shock index/*indeks syok (IS)*
UAP: unstable angina pectoris

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## Ethical Clearance

No: 107/TGL/KEPK FK USU-RSUP HAM/2017 from Research Ethics Committee, Faculty of Medicine Universitas Sumatera Utara.

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